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places; the metacentric chromosome derived from the short arm of chromosome 1 translocated to the short arm of chromosome 2 (Andrews *et al.* 1984), and a chromosome 16 with a deleted short arm. The single X chromosome was abnormal, with a wide positive band in the long arm at q25. Neither the previously reported numerous chromosome 12 involvements, the isochromosome of the long arm of 17, nor the deleted chromosome 22 were seen (Andrews *et al.* 1984). A number of characteristic marker chromosomes was observed but we could not confidently determine which chromosomes had contributed to these extra elements. To reduce the possibility that changes in the modal chromosome number might alter the behaviour of the clones during the course of these experiments, we made our observations on cells that had been grown for less than 16 passages after cloning.

Clones 12 and 13 were shown to form tumours at 2–3 months after inoculation of the cells into nude mice. The tumour formed by clone 12 cells weighed 0·032 g, and the two tumours from clone 13 cells weighed 0·014 and 0·055 g. On sectioning these were found to contain groups of cells with the appearance of carcinoma, loose connective tissue and cuboidal epithelia around cavities, and there were long processes deep in the tumour that had the appearance and staining properties of nerve axons (Figs 3–6). There were also cells with a variety of other morphologies. These tumours resembled those formed by uncloned Tera-2 in nude mice (Jewett, 1978). Unlike the tumours formed by the NTera-2 clones, glandular epithelia were not found, and the tumours also lacked the cells described by histopathologists as 'embryonal carcinoma cells arranged in papillary formation', which are found in primary teratomas.

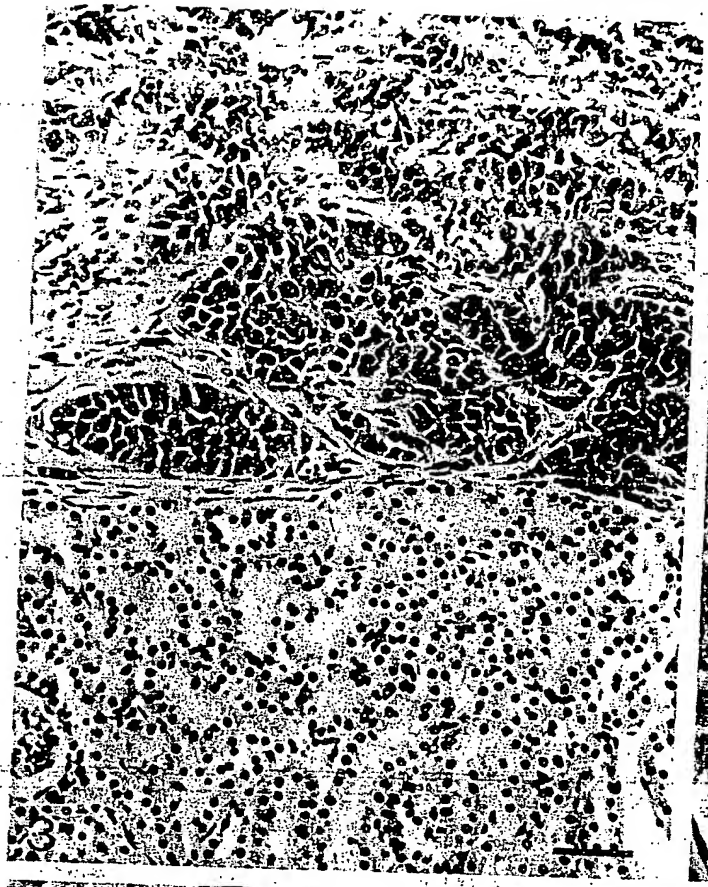
After exposure to retinoic acid in monolayer culture, the cell numbers in the dishes increased approximately fivefold in the next 7–10 days. Measurements of amounts of nuclear DNA in the undifferentiated and treated populations showed that within 8 days the cells transit from a rapidly dividing state, with DNA amounts ranging from $3C$ to $8C$, to a population in which the majority of the cells are arrested in the G_1 phase of the cell cycle displaying $3C$ nuclear DNA amounts (Figs 7–10). In many experiments, the retinoic acid was withdrawn at this time, and the cell number did not increase further during the following 2 months.

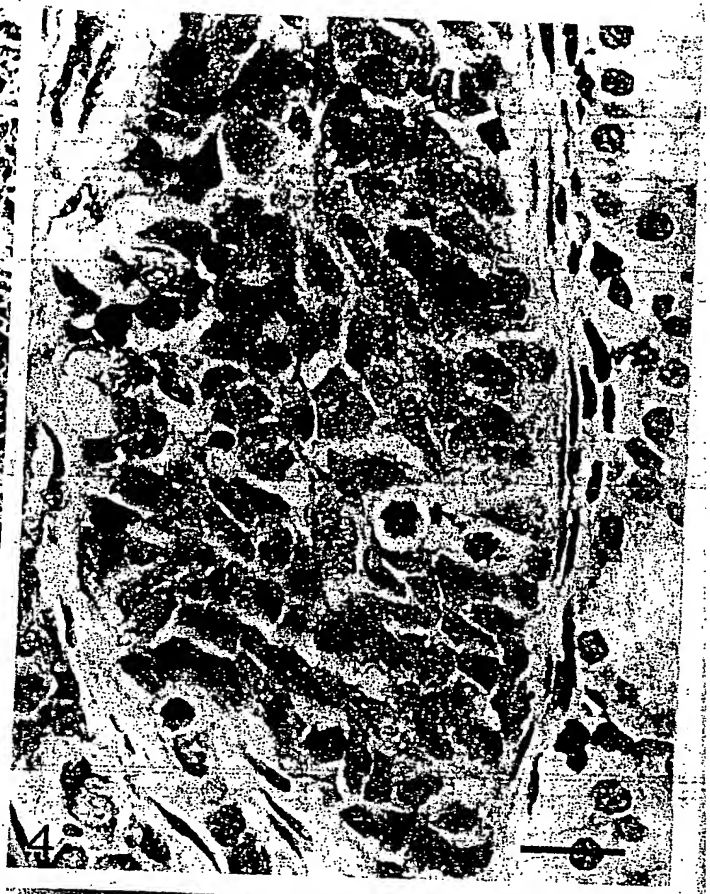
A variety of cell morphologies was seen in the treated cultures. The undifferentiated cells seem to have disappeared from these cultures because their characteristic morphology was not apparent after an extensive search of several hundred retinoic-acid-treated cultures. Further, when the cells were cultured on for several months in the absence of retinoic acid, then in four separate experiments with both clone 12 and clone 13 cells, the undifferentiated morphology did not recur.

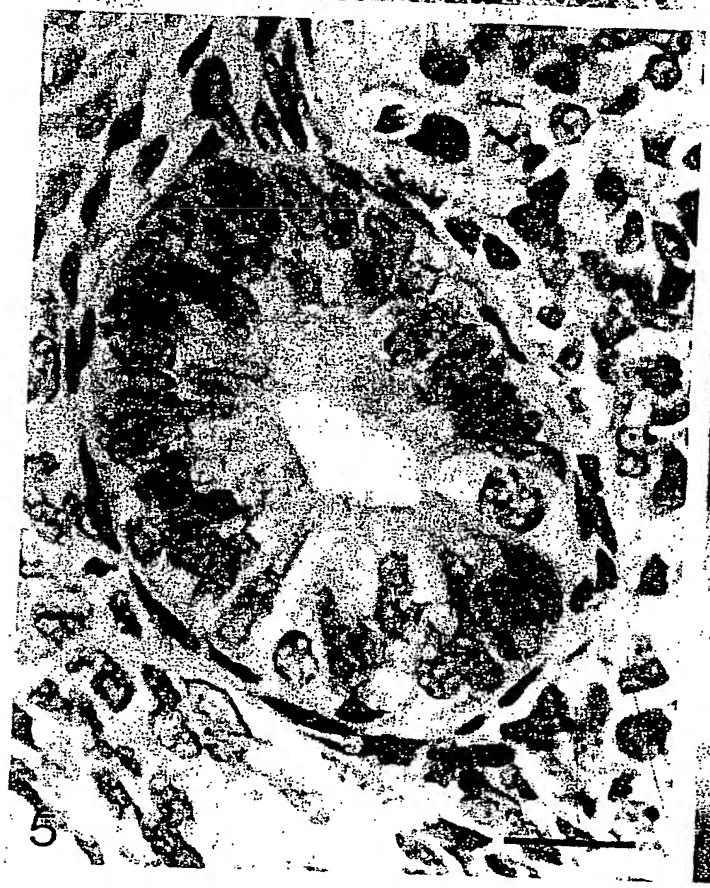
Changes in surface phenotype on differentiation in monolayer culture

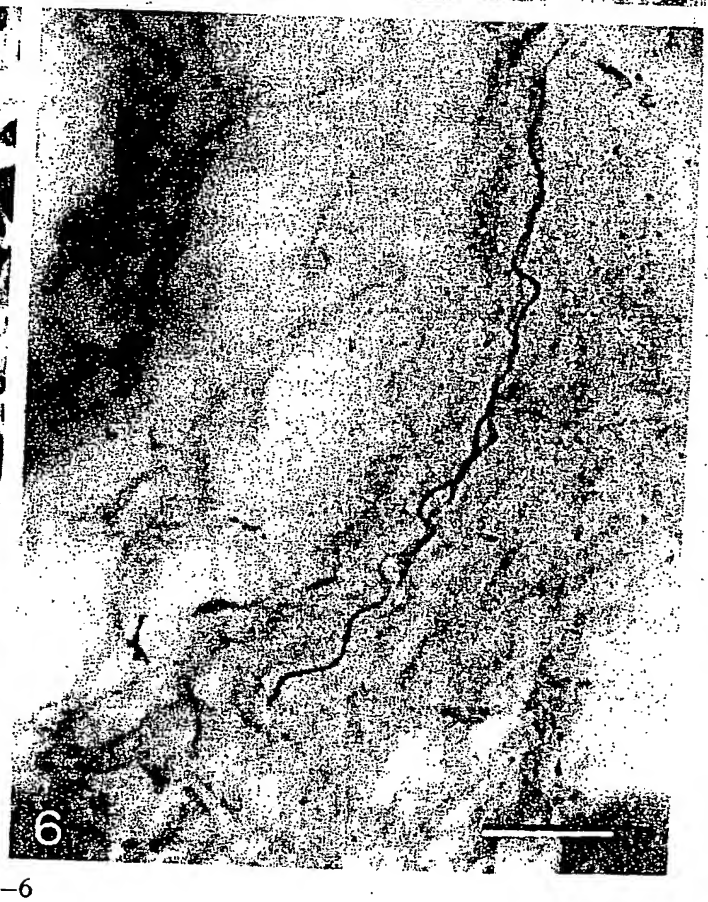
We characterized the phenotype of undifferentiated cell populations and of the

Fig. 2. G-banded karyotype of Tera-2, clone 13: 61, X (abnormal) Y, −1, +del 1, +del 1, +del 1, +t(1;2), +3, +7, +9, +11, +del 16, +21, +mar, +mar, +mar, +mar, +mar, +mar (?12 h). Although there were variations in the marker chromosomes, the other features described in this legend were apparent in each of the 10 karyotypes analysed in detail.









Figs 3–6

differentiated cells formed in monolayer cultures in response to retinoic acid. The antigenic phenotype of the undifferentiated cells was distinct from that of the NTera-2 clones, and there were striking changes in the expression of HLA-A,B,C common determinants during this differentiation.

The expression of HLA-A,B,C common antigens and β_2-microglobulin was studied with monoclonal antibodies to these determinants (W6/32, PA 2·6, anti-β_2). The undifferentiated cell populations rarely reacted with these monoclonal antibodies (Table 2) and a similar result was obtained with MHM5, another monoclonal to HLA-A,B,C common determinants (results not shown). In those experiments in which more than 3 % of the undifferentiated cell populations appeared to react with these antibodies, an independent observer had previously recorded that the populations contained morphologically distinct flat cells. There is therefore a close association between the undifferentiated appearance and the lack of reactivity with these antibodies. The majority of these observations were made by eye, but the rare reactivity of undifferentiated cell populations was also confirmed on FACS. It was found that 2·5 % more cells reacted with W6/32 than with an irrelevant antibody (W3/25) in a sample that was scored as 3 % reactive with W6/32 by eye.

After exposure to retinoic acid for a week, there was a considerable increase in the proportion of cells that reacted with the monoclonal antibodies W6/32, PA 2.6, and the anti-β_2-microglobulin antibody (Table 2). This observation was confirmed on the FACS using the W6/32 antibody reaction to clone 12. This new phenotype was stable following withdrawal of retinoic acid after treatment for 1 week; for example, 1 month after withdrawal, the differentiated clone 13 cells exhibited 97 % reactivity with W6/32, 100 % reactivity with PA 2.6, and 100 % reactivity with the anti-β_2-microglobulin antibody. In another experiment, 4 months after the withdrawal of retinoic acid, 55 % of clone 12 differentiated cells reacted with W6/32. The increase in reactivity with W6/32, PA 2.6, and anti-β_2-microglobulin antibodies was progressive during continuous exposure to retinoic acid: for instance, in one particular experiment, the proportion of clone 12 cells that reacted with W6/32 increased progressively to give 1·3 % reactivity at 2 days, 7 % at 4 days, 26 % at 6 days and 53 % at 9 days. The reactivity of the cells with PA 2.6 and anti-β_2-microglobulin antibodies increased in parallel with W6/32 reactivity in this experiment.

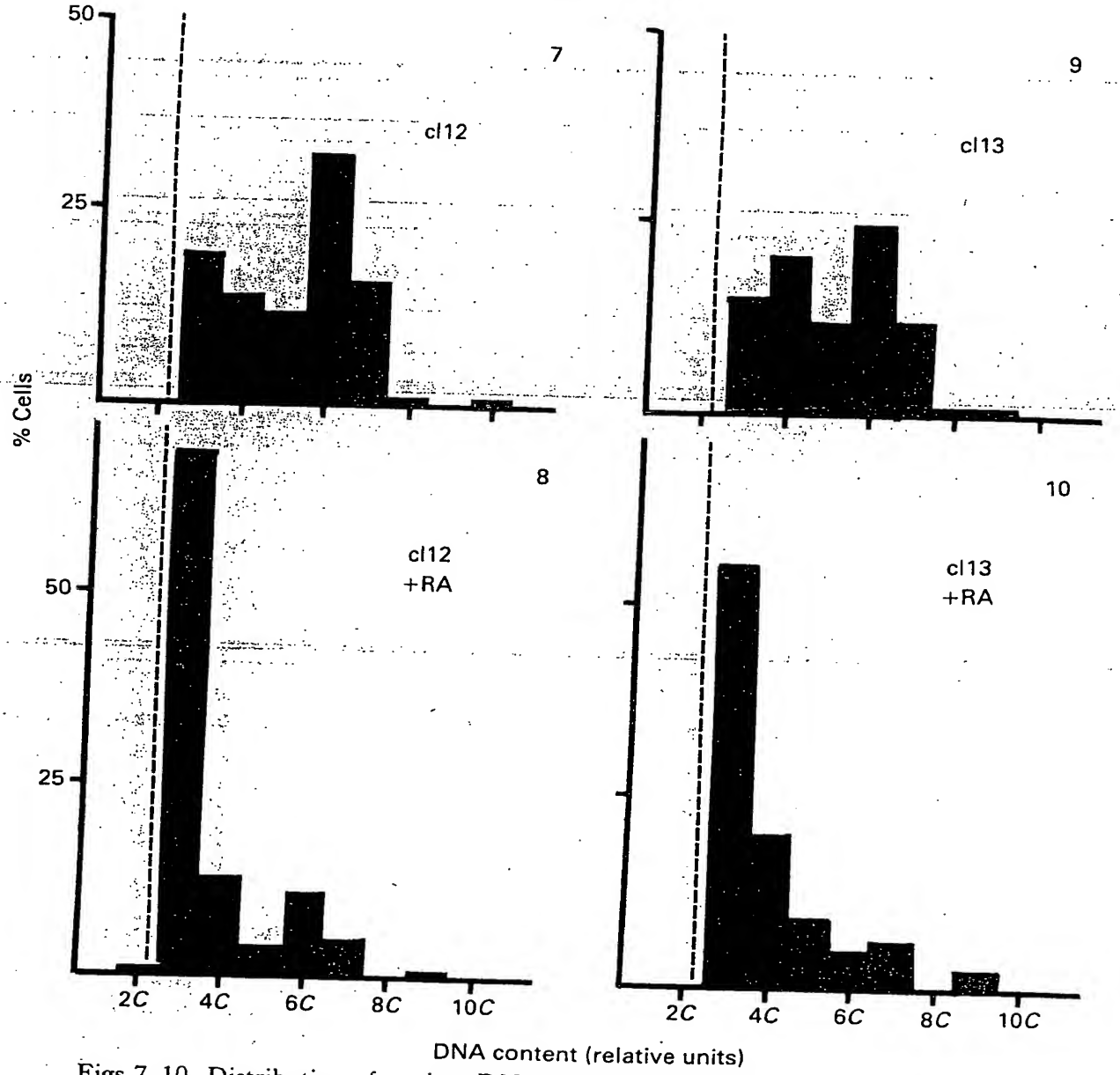
Figs 3–6. Histology of tumours formed by Tera-2 cells grown under the kidney capsule of nude mice.

Fig. 3. Tera-2, clone 13. The tumour principally consists of nests of carcinoma cells in loose connective tissue. Apparently normal mouse kidney tubules occupy the bottom half of the figure. Bar, 50 μm.

Fig. 4. Tera-2, clone 13. A nest of carcinoma cells with a high mitotic index beside kidney tubules along the right of the picture. Bar, 20 μm.

Fig. 5. Tubule within a Tera-2, clone 12 tumour. The simple epithelium is surrounded by connective tissue. Bar, 20 μm.

Fig. 6. Axon-like process within a Tera-2, clone 12 tumour. It is impossible to exclude the possibility that the axon is derived from the surrounding host kidney but it was found deep in the tumour. Holmes' silver stain. Bar, 20 μm.



Figs 7–10. Distribution of nuclear DNA content (Feulgen staining material) in undifferentiated cells and differentiated cells, formed after 8 days retinoic acid (RA) treatment. Each histogram is based on the cytophotometric analysis of approximately 100 cells. The DNA values are expressed in relation to the mean staining values of the control cerebellum cells: these were given the arbitrary value of 2*C* (denoting the diploid DNA content), and a broken line on each figure represents the upper limit of the staining control (2·1*C*).

Fig. 7. Clone 12, undifferentiated.
Fig. 8. Clone 12, differentiated.
Fig. 9. Clone 13, undifferentiated.
Fig. 10. Clone 13, differentiated.

The monoclonal antibody W6/32 was used to immunoprecipitate leucine-labelled material from the differentiated cells and it specifically precipitated a $43 \times 10^3 M_r$ molecule, which is slightly smaller than the expected size of HLA-A,B,C polypeptides.

The majority of the undifferentiated cells of the NTera-2 clones were known to

Table 1. *Reactivities of reagents*

Monoclonal antibody or reagent (original reference)	Major determinant or molecule bearing determinant (reference)
W6/32 (1)	HLA-A,B,C (44*) in association with human or mouse β_2-microglobulin (12*). Weak reaction with HLA alone, none with β_2-microglobulin alone (2,3,4,5). Structural gene on chromosome 6
PA 2.6 (6)	HLA-A,B,C (44*) in association with human β_2-microglobulin (12*) (7)
MHM-5 (8)	HLA-A,B,C (44*) in association with human β_2-microglobulin (12*) (8)
Anti-β_2-microglobulin (9)	β_2-microglobulin (12*) alone or in association with HLA-A,B,C. Structural gene on chromosome 15 (7)
SSEA-3 (10)	3Ga1NAcβ1→3Ga1α1→4Ga1, on glycoproteins and glycolipids of an human teratoma line (2102Ep); on preimplantation mouse embryos up to the 8-cell stage (10,11,12)
SSEA-1 (13)	Ga1β1→4(Fucα1→3)G1cNAc, principally found on glycolipids; on preimplantation mouse embryos from morula stage onwards (14,15,16)
F10.44.2 (17)	87–89 × $10^3 M_r$ sialoglycoproteins on human brain and blood mononulear cells. Structural locus or expression control on chromosome 11 (17,18,19)
F15.42.1 (20)	23·5 × $10^3 M_r$, human Thy-1 (20)
RT 97 (21)	210 × $10^3 M_r$ neurofilament on human brain (21)
BF10 (21)	155 × $10^3 M_r$ neurofilament on human brain (21)
Tetanus toxin	Reacts with gangliosides with decreasing affinity, GT1b,GD1b,GD1a. Reacts with neurons, astrocytes, pancreatic cells, thyroid cells and present on 10th day mouse brain (22,23,24,25,26,27)
F12.A2B5 (28)	Highest affinity for GQ1c gangliosides. Reacts with neurons, some astrocytes, some pre-oligodendrocytes and 'APUD' cells (25,28,29)
GFAP (30)	Glial fibrillary acidic protein. Restricted to glial cells (30)
U13A (31)	On human foetal muscle cultures, reacting with myoblasts, myotubes immature myofibres and adult regenerating myofibres. On neurons in foetal brain cultures (32,33,34,35)
5.1H11 (33)	Distribution similar to U13A
MIN 1 (36)	Astrocytes of human brain cultures and granulocytes (36,37)
O4 (39)	Mouse oligodendrocytes and galactocerebroside positive cells on human brain cultures (35,38,39)
2.3 F9 (40)	Fibronectin (40)

The origin of the reagents and the distribution of their reactivities is described in these references. (1) Williams *et al.* (1977); (2) Barnstable *et al.* (1978); (3) Parham *et al.* (1979); (4) Trowsdale *et al.* (1980); (5) Goodfellow *et al.* (1976); (6) Parham & Bodmer (1978); (7) Brodsky *et al.* (1979); (8) Hildreth (1982); (9) Becton Dickinson; (10) Shevinsky *et al.* (1982); (11) Kannagi *et al.* (1983*a*); (12) Kannagi *et al.* (1983*b*); (13) Solter & Knowles (1978); (14) Gooi *et al.* (1981); (15) Hakomori *et al.* (1981); (16) Kannagi *et al.* (1982); (17) Dalchau *et al.* (1980); (18) Goodfellow *et al.* (1982*a,b*); (19) McKenzie *et al.* (1982); (20) McKenzie & Fabre (1981); (21) Anderton *et al.* (1982); (22) Van Heynigen (1963); (23) Lebdeen & Mellanby (1977); (24) Rogers & Snyder (1981); (25) Eisenbarth *et al.* (1982); (26) Raff *et al.* (1983); (27) Koulakoff *et al.* (1983); (28) Eisenbarth *et al.* (1979); (29) Kasai & Yu (1983); (30) Pruss (1979); (31) J. T. Kemshead; (32) Walsh *et al.* (1983); (33) Walsh *et al.* (1981); (34) Walsh & Ritter (1981); (35) Hurko & Walsh (1983); (36) G. Dickson, personal communication; (37) Kemshead *et al.* (1981); (38) Dickson *et al.* (1983); (39) Somner & Schachner (1981); (40) Walsh *et al.* (1981).

* Molecular weights are given in parenthesis ($\times 10^{-3}$).

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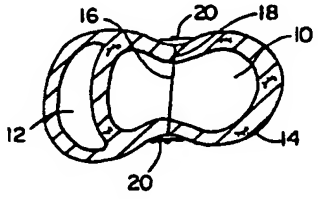

(57) Abstract

This invention is an apparatus for treatment of a failing heart by reducing the wall tension therein. In one embodiment, the apparatus includes a tension member (18) for drawing at least two walls of a heart chamber toward each other. Methods for placing the apparatus on the heart are also provided.

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HEART WALL TENSION REDUCTION APPARATUS AND METHOD

Cross Reference to Related Application

This application is a continuation-in-part of U.S. Application Serial No. 08/778,277, filed January 2, 1997, entitled "HEART WALL TENSION REDUCTION APPARATUS".

Field of the Invention

The present invention pertains to the field of apparatus for treatment of a failing heart. In particular, the apparatus of the present invention is directed toward reducing the wall stress in the failing heart.

Background of the Invention

The syndrome of heart failure is a common course for the progression of many forms of heart disease. Heart failure may be considered to be the condition in which an abnormality of cardiac function is responsible for the inability of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues, or can do so only at an abnormally elevated filling pressure. There are many specific disease processes that can lead to heart failure with a resulting difference in pathophysiology of the failing heart, such as the dilatation of the left ventricular chamber. Etiologies that can lead to this form of failure include

idiopathic cardiomyopathy, viral cardiomyopathy, and ischemic cardiomyopathy.

The process of ventricular dilatation is generally the result of chronic volume overload or specific damage to the myocardium. In a normal heart that is exposed to long term increased cardiac output requirements, for example, that of an athlete, there is an adaptive process of slight ventricular dilation and muscle myocyte hypertrophy. In this way, the heart fully compensates for the increased cardiac output requirements. With damage to the myocardium or chronic volume overload, however, there are increased requirements put on the contracting myocardium to such a level that this compensated state is never achieved and the heart continues to dilate.

The basic problem with a large dilated left ventricle is that there is a significant increase in wall tension and/or stress both during diastolic filling and during systolic contraction. In a normal heart, the adaptation of muscle hypertrophy (thickening) and ventricular dilatation maintain a fairly constant wall tension for systolic contraction. However, in a failing heart, the ongoing dilatation is greater than the hypertrophy and the result is a rising wall tension requirement for systolic contraction. This is felt to be an ongoing insult to the muscle myocyte resulting in

further muscle damage. The increase in wall stress is also true for diastolic filling. Additionally, because of the lack of cardiac output, there is generally a rise in ventricular filling pressure from several physiologic mechanisms. Moreover, in diastole there is both a diameter increase and a pressure increase over normal, both contributing to higher wall stress levels. The increase in diastolic wall stress is felt to be the primary contributor to ongoing dilatation of the chamber.

Prior art treatments for heart failure fall into three generally categories. The first being pharmacological, for example, diuretics. The second being assist systems, for example, pumps. Finally, surgical treatments have been experimented with, which are described in more detail below.

With respect to pharmacological treatments, diuretics have been used to reduce the workload of the heart by reducing blood volume and preload. Clinically, preload is defined in several ways including left ventricular end diastolic pressure (LVEDP), or left ventricular end diastolic volume (LVEDV). Physiologically, the preferred definition is the length of stretch of the sarcomere at end diastole. Diuretics reduce extra cellular fluid which builds in congestive heart failure patients increasing preload conditions. Nitrates, arteriolar vasodilators, angiotensin converting

enzyme inhibitors have been used to treat heart failure through the reduction of cardiac workload through the reduction of afterload. Afterload may be defined as the tension or stress required in the wall of the ventricle during ejection. Inotropes like digoxin are cardiac glycosides and function to increase cardiac output by increasing the force and speed of cardiac muscle contraction. These drug therapies offer some beneficial effects but do not stop the progression of the disease.

Assist devices include mechanical pumps and electrical stimulators. Mechanical pumps reduce the load on the heart by performing all or part of the pumping function normally done by the heart. Currently, mechanical pumps are used to sustain the patient while a donor heart for transplantation becomes available for the patient. Electrical stimulation such as bi-ventricular pacing have been investigated for the treatment of patients with dilated cardiomyopathy.

There are at least three surgical procedures for treatment of heart failure: 1) heart transplant; 2) dynamic cardiomyoplasty; and 3) the Batista partial left ventriculectomy. Heart transplantation has serious limitations including restricted availability of organs and adverse effects of immunosuppressive therapies required following heart transplantation. Cardiomyoplasty includes wrapping the heart with skeletal

muscle and electrically stimulating the muscle to contract synchronously with the heart in order to help the pumping function of the heart. The Batista partial left ventriculectomy includes surgically remodeling the left ventricle by removing a segment of the muscular wall. This procedure reduces the diameter of the dilated heart, which in turn reduces the loading of the heart. However, this extremely invasive procedure reduces muscle mass of the heart.

Summary of the Invention

The present invention pertains to a non-pharmacological, passive apparatus and method for the treatment of a failing heart. The device is configured to reduce the tension in the heart wall. It is believed to reverse, stop or slow the disease process of a failing heart as it reduces the energy consumption of the failing heart, decreases isovolumetric contraction, increases sarcomere shortening during contraction and increases isotonic shortening which in turn increases stroke volume. The device reduces wall tension during diastole and systole.

In one embodiment, the apparatus includes a tension member for drawing at least two walls of the heart chamber toward each other to reduce the radius or area of the heart chamber in at least one cross sectional plane.

The tension member has anchoring members disposed at opposite ends for engagement with the heart or chamber wall.

In another embodiment, the apparatus includes a compression member for drawing at least two walls of a heart chamber toward each other. In one embodiment, the compression member includes a balloon. In another embodiment of the apparatus, a frame is provided for supporting the compression member.

Yet another embodiment of the invention includes a clamp having two ends biased toward one another for drawing at least two walls of a heart chamber toward each other. The clamp includes at least two ends having atraumatic anchoring member disposed thereon for engagement with the heart or chamber wall.

In yet another embodiment, a heart wall tension reduction apparatus is provided which includes a first tension member having two oppositely disposed ends and first and second elongate anchor members. A second tension member can be provided. One of the elongate anchors may be substituted for by two smaller anchors.

In an alternate embodiment of the heart wall tension reduction apparatus, an elongate compression member can be provided. First and second elongate lever members preferably extend from opposite ends of the compression

member. A tension member extends between the first and second lever members.

The compression member of the above embodiment can be disposed exterior to, or internally of the heart. The tension member extends through the chamber or chambers to bias the lever members toward the heart.

In yet another embodiment of a heart wall tension reduction apparatus in accordance with the present invention, a rigid elongate frame member is provided. The frame member can extend through one or more chambers of the heart. One or more cantilever members can be disposed at opposite ends of the frame member. Each cantilever member includes at least one atraumatic pad disposed thereon. The atraumatic pads disposed at opposite ends of the frame member can be biased toward each other to compress the heart chamber.

One method of placing a heart wall tension apparatus or splint on a human heart includes the step of extending a hollow needle through at least one chamber of the heart such that each end of the needle is external to the chamber. A flexible leader is connected to a first end of a tension member. A second end of the tension member is connected to an atraumatic pad. The leader is advanced through the needle from one end of the needle to the other. The leader is further advanced until the second end of the tension member is proximate the heart

and the first end of the tension member is external to the heart. A second atraumatic pad is connected to the first end of the tension member such that the first and second atraumatic pads engage the heart.

An alternate method of placing the heart wall tension reduction apparatus on the heart includes the step of extending a guide member through at least one chamber of the heart such that each end of the guide member is external to the chamber. A tension member for use in this method has at least one lumen extending through at least a portion of the member. The guide member is placed in the lumen. The tension member is advanced over the guide member such that a first end of the tension member is disposed to one side of and external to the heart and a second end of the tension member is disposed to an opposite side of and external to the heart. A first atraumatic pad is connected to one end of the tension member and a second atraumatic pad is connected to the opposite end of the tension member.

Yet another method of placing a heart wall tension apparatus on a heart includes the step of extending a needle having a flexible tension member releasably connected thereto through at least one chamber of the heart such that opposite ends of the tension member are external to the chamber and exposed on opposite sides of the chamber. The needle is removed from the tension

member. Then first and second atraumatic pads are connected to the tension member at opposite ends of the tension member.

Brief Description of the Drawings

Figure 1 is a transverse cross-section of the left and right ventricles of a human heart showing the placement of a splint in accordance with the present invention;

Figure 2 is a transverse cross-section of the left and right ventricles of a human heart showing the placement of a balloon device in accordance with the present invention;

Figure 3 is a transverse cross-section of the left and right ventricles of a human heart showing the placement of an external compression frame structure in accordance with the present invention;

Figure 4 is a transverse cross-section of the left and right ventricles of a human heart showing a clamp in accordance with the present invention;

Figure 5 is a transverse cross-section of the left and right ventricles of a human heart showing a three tension member version of the splint of Figure 1;

Figure 6 is a transverse cross-section of the left and right ventricles of a human heart showing a four tension member version of the splint shown in Figure 1;

Figure 7 is a vertical cross-sectional view of the left ventricle of a human heart showing an alternate version of the splint in accordance with the present invention;

Figure 8 is an end of the splint shown in Figure 7;

Figure 9 is a vertical cross-sectional view of a chamber of a human heart showing another alternative embodiment of the splint in accordance with the present invention;

Figure 10 is a vertical cross-section of a chamber of a human heart showing another alternative configuration of splints in accordance with the present invention;

Figure 11 is a vertical cross-sectional view of a chamber of a human heart showing another embodiment of a splint in accordance with the present invention;

Figure 12 is a vertical cross-sectional view of a chamber of a human heart showing another embodiment of the splint in accordance with the present invention;

Figure 13 is a vertical cross-sectional view of a chamber of a human heart showing a compression member version of the splint in accordance with the present invention;

Figure 14 is a vertical cross-sectional view of a chamber of a human heart showing another version of the splint shown in Figure 13;

Figure 15 is a vertical cross-sectional view of a chamber of a human heart showing a frame member version of the splint in accordance with the present invention;

Figure 16 is an end view of the splint of Figure 15;

Figure 17 is a vertical cross-section of the left ventricle and atrium, the left ventricle having scar tissue;

Figure 18 is a vertical cross-section of the heart of Figure 7 showing the splint of Figure 1 drawing the scar tissue toward the opposite wall of the left ventricle;

Figure 19 is a vertical cross-section of the left ventricle and atrium of a human heart showing a version of the splint of Figure 1 having an elongate anchor bar;

Figure 20 is a side view of an undeployed hinged anchor member;

Figure 21 is a side view of a deployed hinged anchor member of Figure 10;

Figure 22 is a cross-sectional view of an captured ball anchor member;

Figure 23 is a perspective view of a cross bar anchor member;

Figure 24 is a vertical cross-sectional view of a chamber of a human heart showing a needle used for placement of splint in accordance with the present invention;

Figure 25 is a view of the heart and needle of Figure 24 showing a tension member being placed in the heart;

Figure 26 is a view of the heart shown in Figure 24 wherein oppositely disposed anchor pads are being joined by a tension member;

Figure 27 is a view of the heart of Figure 24, wherein two oppositely disposed anchor pads have been joined by two tension members;

Figure 28 is a view of a tension member having a lumen extending therethrough;

Figure 29 is a view of a tension member having lumens extending therethrough;

Figure 30 is a vertical cross-sectional view of a chamber of the heart and two pads, and a needle extending therethrough;

Figure 31 is a vertical cross-sectional view of a chamber of the heart showing a guidewire extending therethrough;

Figure 32 is a view of the heart of Figure 31, and two pads, and a guidewire extending therethrough;

Figure 33 is a vertical cross-sectional view of a chamber of the heart showing a needle connected to a tension member being inserted into the chamber;

Figure 34 is a vertical cross-sectional view of a chamber of a heart showing two anchors connected by a tension member;

Figure 35 is a vertical cross-sectional view of a chamber of the heart, showing a band surrounding the heart;

Figure 36 is a idealized cylindrical model of a left ventricle of a human heart;

Figure 37 is a splinted model of the left ventricle of Figure 14;

Figure 38 is a transverse cross-sectional view of Figure 15 showing various modeling parameters;

Figure 39 is a transverse cross-section of the splinted left ventricle of Figure 15 showing a hypothetical force distribution; and

Figure 40 is a second transverse cross-sectional view of the model left ventricle of Figure 15 showing a hypothetical force distribution.

Detailed Description of the Invention

Referring now to the drawings wherein like reference numerals refer to like elements throughout the several views, Figure 1 shows a transverse cross-section of a left ventricle 10 and a right ventricle 12 of a human heart 14. Extending through the left ventricle is a splint 16 including a tension member 18 and oppositely

disposed anchors 20. Splint 16 as shown in Figure 1 has been positioned to draw opposite walls of left ventricle 10 toward each other to reduce the "radius" of the left ventricular cross-section or the cross-sectional area thereof to reduce left ventricular wall stresses. It should be understood that although the splint 16 and the alternative devices disclosed herein are described in relation to the left ventricle of a human heart, these devices could also be used to reduce the radius or cross-sectional area of the other chambers of a human heart in transverse or vertical directions, or at an angle between the transverse and vertical.

Figure 2 discloses an alternate embodiment of the present invention, wherein a balloon 200 is deployed adjacent the left ventricle. The size and degree of inflation of the balloon can be varied to reduce the radius or cross-sectional area of left ventricle 10 of heart 14.

Figure 3 shows yet another alternative embodiment of the present invention deployed with respect to left ventricle 10 of human heart 14. Here a compression frame structure 300 is engaged with heart 14 at atraumatic anchor pads 310. A compression member 312 having an atraumatic surface 314 presses against a wall of left ventricle 10 to reduce the radius or cross-sectional area thereof.

Figure 4 is a transverse cross-sectional view of human heart 14 showing yet another embodiment of the present invention. In this case a clamp 400 having atraumatic anchor pads 410 biased toward each other is shown disposed on a wall of left ventricle 10. Here the radius or cross-sectional area of left ventricle 10 is reduced by clamping off the portion of the wall between pads 410. Pads 410 can be biased toward each other and/or can be held together by a locking device.

Each of the various embodiments of the present invention disclosed in Figures 1-4 can be made from materials which can remain implanted in the human body indefinitely. Such biocompatible materials are well-known to those skilled in the art of clinical medical devices.

Figure 5 shows an alternate embodiment of the splint of Figure 1 referred to in Figure 5 by the numeral 116. The embodiment 116 shown in Figure 5 includes three tension members 118 as opposed to a single tension member 18 as shown in Figure 1. Figure 6 shows yet another embodiment of the splint 216 having four tension members 218. It is anticipated that in some patients, the disease process of the failing heart may be so advanced that three, four or more tension members may be desirable to reduce the heart wall stresses more substantially than

possible with a single tension member as shown in Figure 1.

Figure 7 is a partial vertical cross-section of human heart 14 showing left ventricle 10. In Figure 7, another splint embodiment 316 is shown having a tension member 318 extending through left ventricle 10. On opposite ends of tension member 318 are disposed elongate anchors or pads 320. Figure 8 is an end view of tension member 318 showing elongate anchor 320.

Figure 9 shows another embodiment of a splint 416 disposed in a partial vertical cross-section of human heart 14. Splint 416 includes two elongate anchors or pads 420 similar to those shown in Figures 7 and 8. In Figure 9, however, two tension members 418 extend through left ventricle 10 to interconnect anchors 420 on opposite sides of heart 14.

Figure 10 is a vertical cross section of heart 14 showing left ventricle 10. In this case, two splints 16 are disposed through left ventricle 10 and vertically spaced from each other to resemble the configuration of Figure 9.

Figure 11 is a vertical cross sectional view of the left ventricle of heart 14. Two alternate embodiment splints 516 are shown extending through left ventricle 10. Each splint 516 includes two tension members 518 interconnecting two anchors or pads 520.

Figure 12 is yet another vertical cross sectional view of left ventricle 10 of heart 14. An alternate embodiment 616 of the splint is shown extending through left ventricle 10. Splint 616 includes an elongate anchor pad 620 and two shorter anchors or pads 621. Splint 616 includes two tension members 618. Each tension member 618 extends between anchors 620 and respective anchors 621.

Figure 13 is a vertical cross sectional view of left ventricle 10 of heart 14. A splint 50 is shown disposed on heart 14. Splint 50 includes a compression member 52 shown extending through left ventricle 10. Opposite ends of compression member 52 are disposed exterior to left ventricle 10. Lever members 54 extend from each end of compression member 52 upwardly along the exterior surface of ventricle 10. A tension member 56 extends between lever members 54 to bias lever members 54 toward heart 14 to compress chamber 10. Compression member 52 should be substantially rigid, but lever members 54 and to some degree compression member 52 should be flexible enough to allow tension member 56 to bias lever members 54 toward heart 14. Alternately, lever members 54 could be hinged to compression member 52 such that lever members 54 could pivot about the hinge when biased toward heart 14 by tension member 56.

Figure 14 shows an alternate embodiment 156 of the splint shown in Figure 13. In this case lever members 154 are longer than members 54 as compression member 152 of splint 150 has been disposed to the exterior of left ventricle 10.

Figure 15 is a vertical cross sectional view of left ventricle 10 of heart 14. An alternate embodiment 250 of the splint is shown on heart 14. A preferably relatively rigid frame member 256 extends through ventricle 10. Disposed on opposite ends of frame 250 are cantilever member 254. Disposed on cantilever members 254 are atraumatic pads 258. Cantilever members 254 can be positioned along frame member 256 such that atraumatic pads 258 press against heart 14 to compress chamber 10. Figure 16 is an end view of frame member 256 showing cantilever members 254 and pads 258.

It should be understood that each of the embodiments described above should be formed from suitable biocompatible materials known to those skilled in the art. The tension members can be formed from flexible or relatively more rigid material. The compression members and frame member should be formed from generally rigid material which may flex under load, but generally hold its shape.

Figure 17 is a partial vertical cross-section of human heart 14 showing left ventricle 10 and left atrium

22. As shown in Figure 7, heart 14 includes a region of scar tissue 24 associated with an aneurysm or ischemia. As shown in Figure 7, the scar tissue 24 increases the radius or cross-sectional area of left ventricle 10 in the region affected by the scar tissue. Such an increase in the radius or cross-sectional area of the left ventricle will result in greater wall stresses on the walls of the left ventricle.

Figure 18 is a vertical cross-sectional view of the heart 14 as shown in Figure 7, wherein a splint 16 has been placed to draw the scar tissue 24 toward an opposite wall of left ventricle 10. As a consequence of placing splint 16, the radius or cross-sectional area of the left ventricle affected by the scar tissue 24 is reduced. The reduction of this radius or cross-sectional area results in reduction in the wall stress in the left ventricular wall and thus improves heart pumping efficiency.

Figure 19 is a vertical cross-sectional view of left ventricle 10 and left atrium 22 of heart 14 in which a splint 16 has been placed. As shown in Figure 9, splint 16 includes an alternative anchor 26. The anchor 26 is preferably an elongate member having a length as shown in Figure 9 substantially greater than its width (not shown). Anchor bar 26 might be used to reduce the radius or cross-sectional area of the left ventricle in an instance where there is generalized enlargement of left

ventricle 10 such as in idiopathic dilated cardiomyopathy. In such an instance, bar anchor 26 can distribute forces more widely than anchor 20.

Figures 20 and 21 are side views of a hinged anchor 28 which could be substituted for anchors 20 in undeployed and deployed positions respectively. Anchor 28 as shown in Figure 20 includes two legs similar to bar anchor 26. Hinged anchor 28 could include additional legs and the length of those legs could be varied to distribute the force over the surface of the heart wall. In addition there could be webbing between each of the legs to give anchor 28 an umbrella-like appearance. Preferably the webbing would be disposed on the surface of the legs which would be in contact with the heart wall.

Figure 22 is a cross-sectional view of a capture ball anchor 30. Capture ball anchor 30 can be used in place of anchor 20. Capture ball anchor 30 includes a disk portion 32 to distribute the force of the anchor on the heart wall, and a recess 34 for receiving a ball 36 affixed to an end of tension member 18. Disk 32 and recess 34 include a side groove which allows tension member 38 to be passed from an outside edge of disk 32 into recess 34. Ball 36 can then be advanced into recess 34 by drawing tension member 18 through an opening 38 in recess 34 opposite disk 32.

Figure 23 is a perspective view of a cross bar anchor 40. The cross bar anchor 40 can be used in place of anchors 20. The anchor 40 preferably includes a disk or pad portion 42 having a cross bar 44 extending over an opening 46 in pad 42. Tension member 18 can be extended through opening 46 and tied to cross bar 42 as shown.

In use, the various embodiments of the present invention are placed in or adjacent the human heart to reduce the radius or cross-section area of at least one chamber of the heart. This is done to reduce wall stress or tension in the heart or chamber wall to slow, stop or reverse failure of the heart. In the case of the splint 16 shown in Figure 1, a canula can be used to pierce both walls of the heart and one end of the splint can be advanced through the canula from one side of the heart to the opposite side where an anchor can be affixed or deployed. Likewise, an anchor is affixed or deployed at the opposite end of splint 16.

Figure 24 is a vertical cross-sectional view of a chamber 10 of a heart 14. A needle 60 having a stylet inserted therethrough is inserted through chamber 10. Figure 25 shows needle 60 disposed in heart 40 as shown in Figure 24. In Figure 25, stylet 62 has been removed. A tension member 64 having a flexible leader 66 attached to one end of tension member 64, is threaded through needle 60 and an anchor 68.

As shown in Figure 25, tension member 64 includes a generally elongate cylindrical shaft 70 having two generally cylindrical ends 72. Ends 72 preferably have a greater diameter than shaft 70. Also shown in Figure 25 is a perspective view of anchor 68 showing an opening 73 extending through anchor 68. Opening 73 includes a first cylindrically shaped opening 74 extending entirely through anchor 68. The diameter of opening 74 is preferably slightly greater than the diameter of end 72 of tension member 64. A groove 76 having a width preferably slightly greater than that of shaft 70 of tension member 64 extends from opening 74 to a generally cylindrical opening 78. Generally cylindrical opening 78 has a diameter approximately equal to end 72. Unlike opening 74, however, opening 78 includes a reduced base opening 80 which has a width approximately equal to that of groove 76. The width of the opening 80 is also less than the diameter of end 72 of tension member 64.

It can be appreciated that tension member 64 can be advanced through opening 74 until shaft 70 is disposed therein. Shaft 70 can be then slid transversely through groove 76. Tension member 64 can then be advanced further through opening 73 until end portion 72 enters opening 78 and seats against base 80.

Figure 26 shows the view of heart 14 shown in Figure 25. Needle 60 has been removed from heart 14. Tension

member 64 has been advanced into chamber 10 and anchor 68 connected thereto is engaging the heart wall. Leader 66 has been advanced through yet another anchor 68 disposed on the opposite side of heart 14.

Figure 27 is a view of heart 14 of Figure 26. Two tension member 64 have been advanced through chamber 10. Each tension member has been seated in respective opening 78 against respective bases 80 to form a splint in a configuration such as that shown in Figure 9.

It can be appreciated that each of the other tension member splints configurations can be placed on the heart in a similar manner. It can also be appreciated that anchors 68 could initially be held against the heart and needle 60 advanced through anchors 68 and chamber 10 prior to extending leader 66 through the needle.

Figure 28 is a perspective view of a tension member 164 in accordance with the present invention. Tension member 164 is similar to tension member 64 described above in that it has an elongate, generally cylindrical shaft 170 and generally cylindrical ends 172. A lumen, however, extends longitudinally through tension member 164 along axis A.

Figure 29 is a perspective view of yet another embodiment of the tension member 264. Tension member 264, is similar to tension member 164, and includes an elongate cylindrical shaft 270 and cylindrical ends 272.

Lumens 282, however, extend through ends 272 aligned along axis B.

Figure 30 is a vertical, cross-sectional view of left ventricle 10 of heart 14. Anchors 68 have been placed on opposite sides of heart 14. A needle 160 extends through the lumen of tension member 164, left ventricle 10 and openings 73 in anchors 68. It can be appreciated that tension member 64 can be advanced through anchors 68 and left ventricle 10 and be seated within openings 78 as described above with respect to tension member 64.

Figure 31 is a vertical, cross-sectional view of left ventricle 10 of heart 14. A needle 60 has been advanced through the wall of left ventricle 10 and a guidewire 162 has been advanced through needle 60.

Figure 32 is the same view of heart 14 as shown in Figure 32. Needle 60, however, has been removed from heart 14 while guidewire 162 remains in position. Anchors 68 have been placed on guidewire 162, on opposite sides of left ventricle 10. Tension member 264 has been threaded onto guidewire 162 through lumens 282. It can be appreciated that as discussed above with respect to tension member 164 above, tension member 264 can be advanced through left ventricle 10 such that ends 272 of tension member 264 seat in respective openings 78 against base 80.

Figure 33 is a vertical, cross-sectional view of left ventricle 10 of heart 14. In Figure 34, flexible tension member 364 has been connected to a needle 360. Needle 360 is shown being advanced into left ventricle 10 through a ventricle wall.

Figure 34 is the same view of heart 14 as shown in Figure 33 except that tension member 364 has been advanced entirely through left ventricle 10 and anchors 68. Knots 384 have been tied at the ends of tension member 364 to prevent the ends of tension member 364 from passing through opening 73 of anchors 68.

It can be appreciated that the methods described above to advance the tension members through the ventricles can be repeated to advance the desired number of tension members through the ventricle for a particular configuration. The length of the tension members can be determined based upon the size and condition of the patient's heart. It should also be noted that although the left ventricle has been referred to here for illustrative purposes, that the apparatus and methods of this invention can also be used to splint multiple chambers of a patient's heart as well as the right ventricle or either atrium.

Figure 35 is a vertical cross-section of left ventricle 10 of heart 14. Disposed about heart 14 is a band 716. Band 716 is shown as being sized relative to

the heart such that the heart's radius or cross-sectional area in a plane parallel to the length of the band is reduced relative to the radius at that location prior to placement of the band on the heart. The length of the heart perpendicular to the band is also increased. The band may be formed from a continuous ribbon of elastomeric material or from other biocompatible materials which are sufficiently strong to provide the desired effect of heart radius reduction and lengthening.

Figure 36 is a view of a cylinder or idealized heart chamber 48 which is used to illustrate the reduction of wall stress in a heart chamber as a result of deployment of the splint in accordance with the present invention. The model used herein and the calculations related to this model are intended merely to illustrate the mechanism by which wall stress is reduced in the heart chamber. No effort is made herein to quantify the actual reduction which would be realized in any particular in vivo application.

Figure 37 is a view of the idealized heart chamber 48 of Figure 36 wherein the chamber has been splinted along its length L such that a "figure eight" cross-section has been formed along the length thereof. It should be noted that the perimeter of the circular transverse cross-section of the chamber in Figure 36 is equal to the perimeter of the figure eight transverse

cross-section of Figure 37. For purposes of this model, opposite lobes of the figure in cross-section are assumed to be mirror images.

Figure 38 shows various parameters of the Figure 1 cross-section of the splinted idealized heart chamber of Figure 37. Where ℓ is the length of the splint between opposite walls of the chamber, R_2 is the radius of each lobe, θ is the angle between the two radii of one lobe which extends to opposite ends of the portion of the splint within chamber 48 and h is the height of the triangle formed by the two radii and the portion of the splint within the chamber 48 (R_1 is the radius of the cylinder of Figure 36). These various parameters are related as follows:

$$h = R_2 \text{ COS } (\theta/2)$$

$$\ell = 2 R_2 \text{ SIN } (\theta/2)$$

$$R_2 = R_1\pi \; / \; (2\pi - \theta)$$

From these relationships, the area of the figure eight cross-section can be calculated by:

$$A_2 = 2\pi(R_2)^2 \; (1-\theta/2\pi) + h\ell$$

Where chamber 48 is unsplinted as shown in Figure 36 A_1, the original cross-sectional area of the cylinder is equal to A_2 where $\theta = 180°$, $h = 0$ and $\ell = 2R_2$. Volume equals A_2 times length L and circumferential wall tension equals pressure within the chamber times R_2 times the length L of the chamber.

Thus, for example, with an original cylindrical radius of four centimeters and a pressure within the chamber of 140 mm of mercury, the wall tension T in the walls of the cylinder is 104.4 newtons. When a 3.84 cm splint is placed as shown in Figures 37 and 38 such that ℓ = 3.84 cm, the wall tension T is 77.33 newtons.

Figures 39 and 40 show a hypothetical distribution of wall tension T and pressure P for the figure eight cross-section. As θ goes from 180° to 0°, tension T_s in the splint goes from 0 to a 2T load where the chamber walls carry a T load.

In yet another example, assuming that the chamber length L is a constant 10 cm, the original radius R_1 is 4 cm, at a 140 mmHg the tension in the walls is 74.7 N. If a 4.5 cm splint is placed such that ℓ = 4.5 cm, the wall tension will then be 52.8 N.

It will be understood that this disclosure, in many respects, is only illustrative. Changes may be made in details, particularly in matters of shape, size, material, and arrangement of parts without exceeding the scope of the invention. Accordingly, the scope of the invention is as defined in the language of the appended claims.

What is claimed is:

1. A heart wall tension reduction apparatus, comprising:

a first tension member having two oppositely disposed ends; and

first and second anchor members, the first anchor member being disposed proximate one end of the tension member and the second anchor member being disposed proximate the opposite end of the tension member.

2. The heart wall tension reduction apparatus in accordance with claim 1, wherein the first anchor member comprises a first pad having a length and a width, and the second anchor member comprises a second pad having a length and a width, and the first and second pad each have two lengthwise ends.

3. The heart wall tension reduction apparatus in accordance with claim 2, wherein the length of the pads is greater than the width of the respective pad.

4. A heart wall tension reduction apparatus in accordance with claim 2, further comprising a second tension member having two oppositely disposed ends; and one lengthwise end of the first pad is disposed proximate one end of the first tension member, and the opposite

lengthwise end of the first pad is disposed proximate one end of the second tension member, and one lengthwise end of the second pad is disposed proximate the end of the first tension member opposite the first pad, and the opposite lengthwise of the second pad is disposed proximate the end of the second tension member opposite the first pad.

5. The heart wall tension reduction apparatus in accordance with claim 4, wherein the length of the first pad is greater than the width of the respective pads.

6. The heart wall tension reduction apparatus in accordance with claim 1, further comprising a third anchor member, and a second tension member having two oppositely disposed ends; wherein the third anchor member is disposed proximate one end of the second tension member and the first anchor member is disposed proximate the opposite end of the second tension member.

7. The heart wall tension reduction apparatus in accordance with claim 6, wherein the first anchor member comprises a first pad having a length and width, and the second anchor member comprises a second pad having a length and a width, and the first and second pads each have two lengthwise ends.

8. The heart wall tension reduction apparatus in accordance with claim 7, wherein the length of the pads is greater than the width of the respective pad.

9. The heart wall tension reduction apparatus, comprising:

an elongate compression member having first and second ends;

first and second elongate lever members, the first lever member extending from the first end of the compression member, and the second lever member extending from the second end of the compression member; and

a tension member extending between the first and second lever members.

10. A heart wall tension reduction apparatus in accordance with claim 9, wherein each of the two lever members have two ends, one end of each of the two lever members is disposed proximate the compression member, the other end of each of the two lever members is disposed remotely from the compression member, and the tension member is disposed closer to the ends of the two lever members disposed proximate the compression member than the ends disposed remotely therefrom.

11. A method of disposing a heart wall tension reduction apparatus on a human heart having a plurality of chambers, comprising the steps of:

providing a heart wall tension reduction apparatus including an elongate compression member having first and second ends; first and second elongate lever members, the first lever member extending from the first end of the compression member, and the second lever member extending from the second end of the compression member; a tension member extending between the first and second lever members; and

extending the tension member through at least one chamber of the human heart.

12. A method in accordance with claim 11, further comprising the step of disposing the compression member external to the heart.

13. The method in accordance with claim 11, further comprising the step of disposing at least a portion of the compression member within the heart.

14. A heart wall tension reduction apparatus, comprising:

a rigid elongate frame member having a first end and a second end;

a first cantilever member disposed at the first end of the frame member; and

a second cantilever member disposed at the second end of the frame member.

15. A heart wall tension reduction apparatus in accordance with claim 14, wherein each cantilever member includes at least one atraumatic pad disposed thereon.

16. A heart wall tension reduction apparatus in accordance with claim 14, further comprising a plurality of cantilever members disposed at the first and second ends of the frame member.

17. The method of placing a heart wall tension reduction apparatus on a human heart having a plurality of chambers, comprising the steps of:

extending a hollow needle through at least one chamber of the heart such that each end of the needle is external to the chamber;

providing a tension member having first and second ends, and a flexible leader connected to the first end of the tension member;

connecting the second end of the tension member to a first atraumatic pad;

advancing the leader through the needle from one end of the needle to the other;

further advancing the leader until the first atraumatic pad engages the heart and the first end of the tension member is external to the at least one chamber; and

connecting the first end of the tension member to a second atraumatic pad such that the second atraumatic pad engages the heart.

18. The method in accordance with claim 17, wherein the length of the tension member is such that the at least one chamber of the heart is compressed between the first and second atraumatic pads.

19. The method of placing a heart wall tension reduction apparatus on a heart having a plurality of chambers, comprising the steps of:

extending a guide member through at least one chamber of the heart such that each end of the guide member is external to the chamber;

providing a tension member having at least one lumen extending therethrough;

extending a portion of the guide member through the lumen;

advancing the tension member on the guide member such that a first end of the tension member is disposed to one side of and external to the at least one chamber, and a second end of the tension member is disposed to an opposite side of and external to the at least one chamber; and

connecting a first atraumatic pad to the first end and a second atraumatic pad to the second end of the tension member.

20. The method in accordance with claim 19, wherein the length of the tension member is such that the at least one chamber of the heart is compressed between the first and second atraumatic pads.

21. A method in accordance with claim 19, wherein the guide member includes a needle.

22. The method in accordance with claim 19, wherein the guide member includes a guidewire.

23. The method of placing a heart wall tension reduction apparatus on a heart having a plurality of chamber comprising the steps of:

extending a needle having a flexible tension member releasably connected thereto through at least one chamber

of the heart such that opposite ends of the tension member are external to the chamber and exposed on opposite sides thereof;

removing the needle from the tension member; and

connecting a first atraumatic pad to one end of the tension member and a second atraumatic pad to the opposite end of the tension member.

24. A method in accordance with claim 23, wherein the length of the tension member is such that the at least one chamber of the heart is compressed between the first and second atraumatic pads.

Fig.1

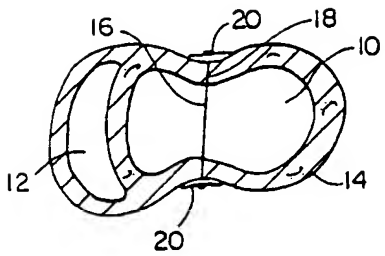


Fig.2

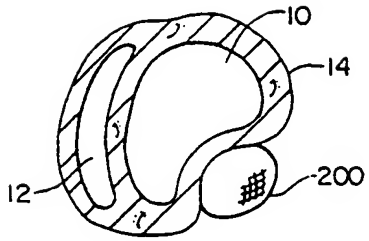


Fig.3

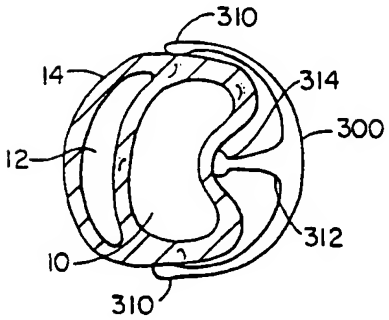


Fig.4

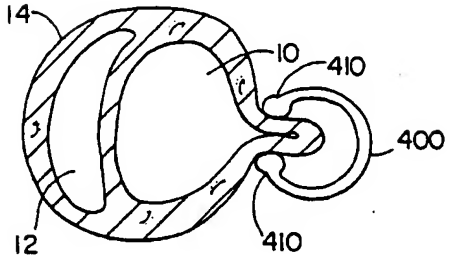


Fig.5

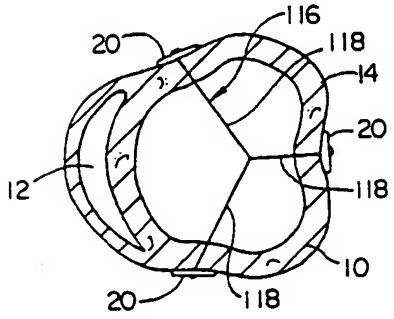


Fig.6

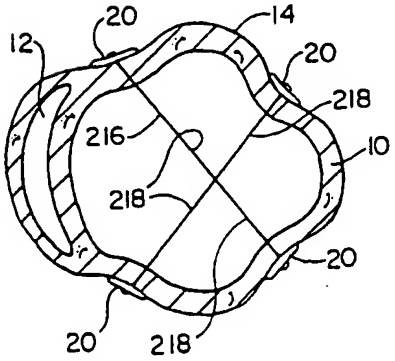


Fig.8

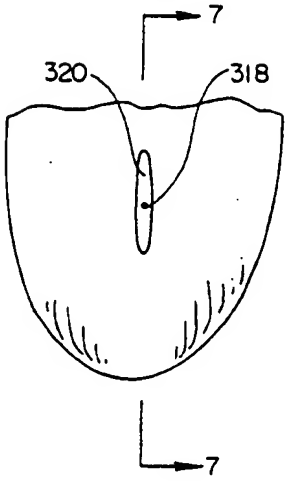


Fig.7

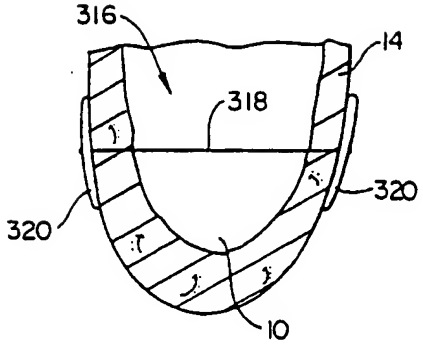


Fig.9

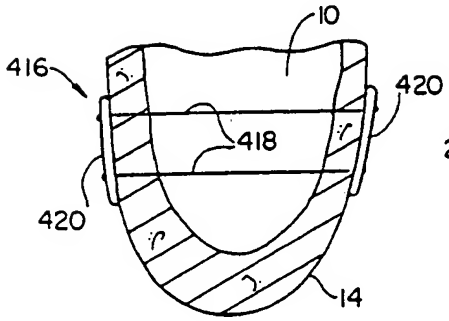


Fig.10

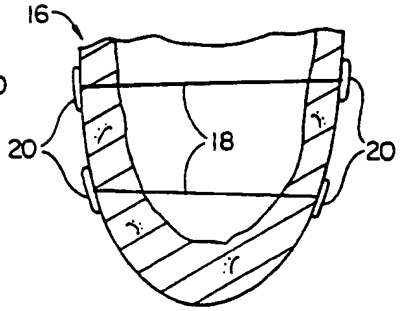


Fig.11

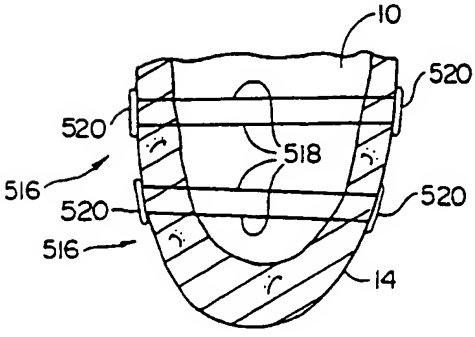


Fig.12

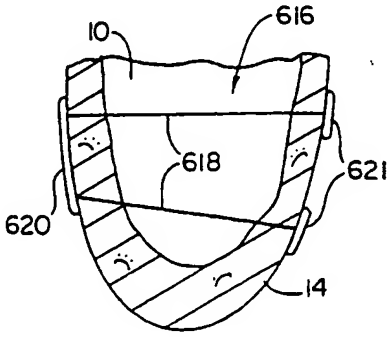


Fig.13

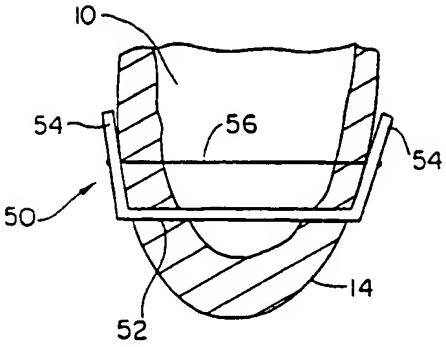


Fig.14

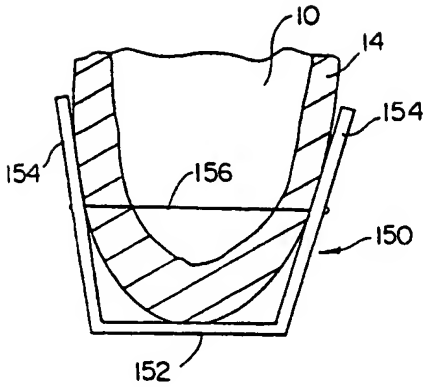


Fig.15

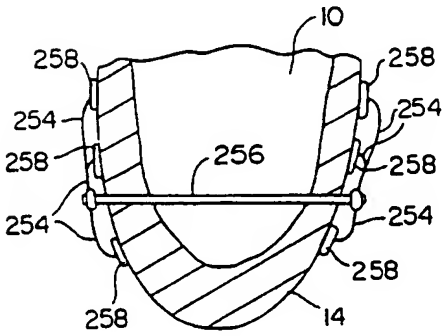


Fig.16

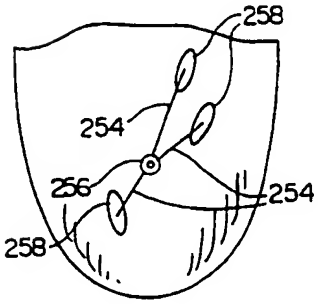


Fig.17

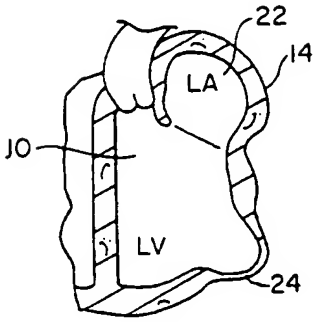


Fig.18

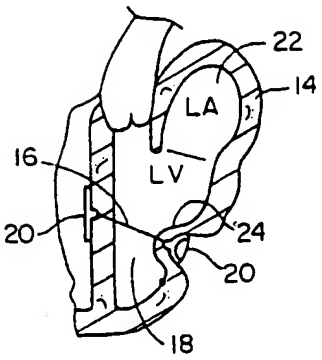
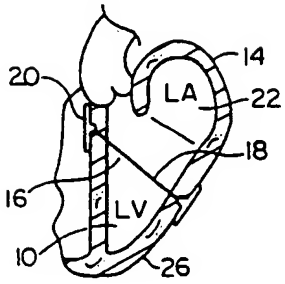


Fig.19



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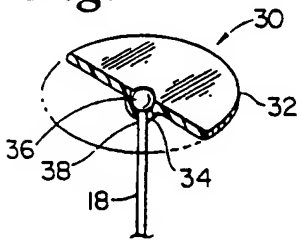


Fig. 23

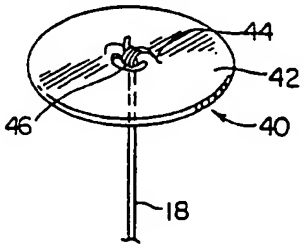


Fig. 20

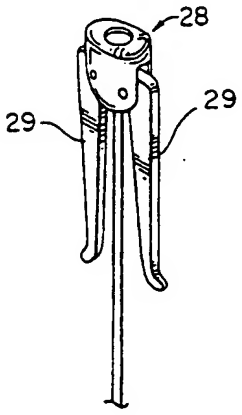


Fig. 21

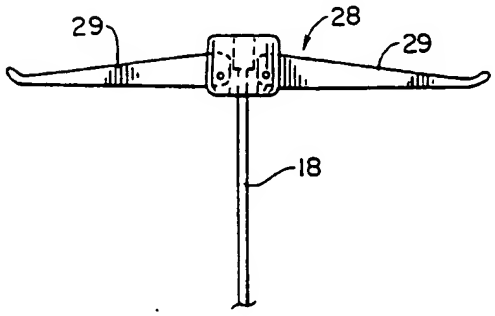
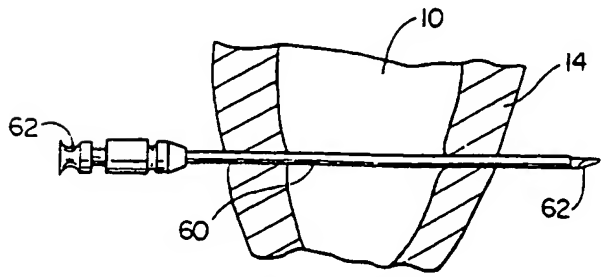


Fig. 24



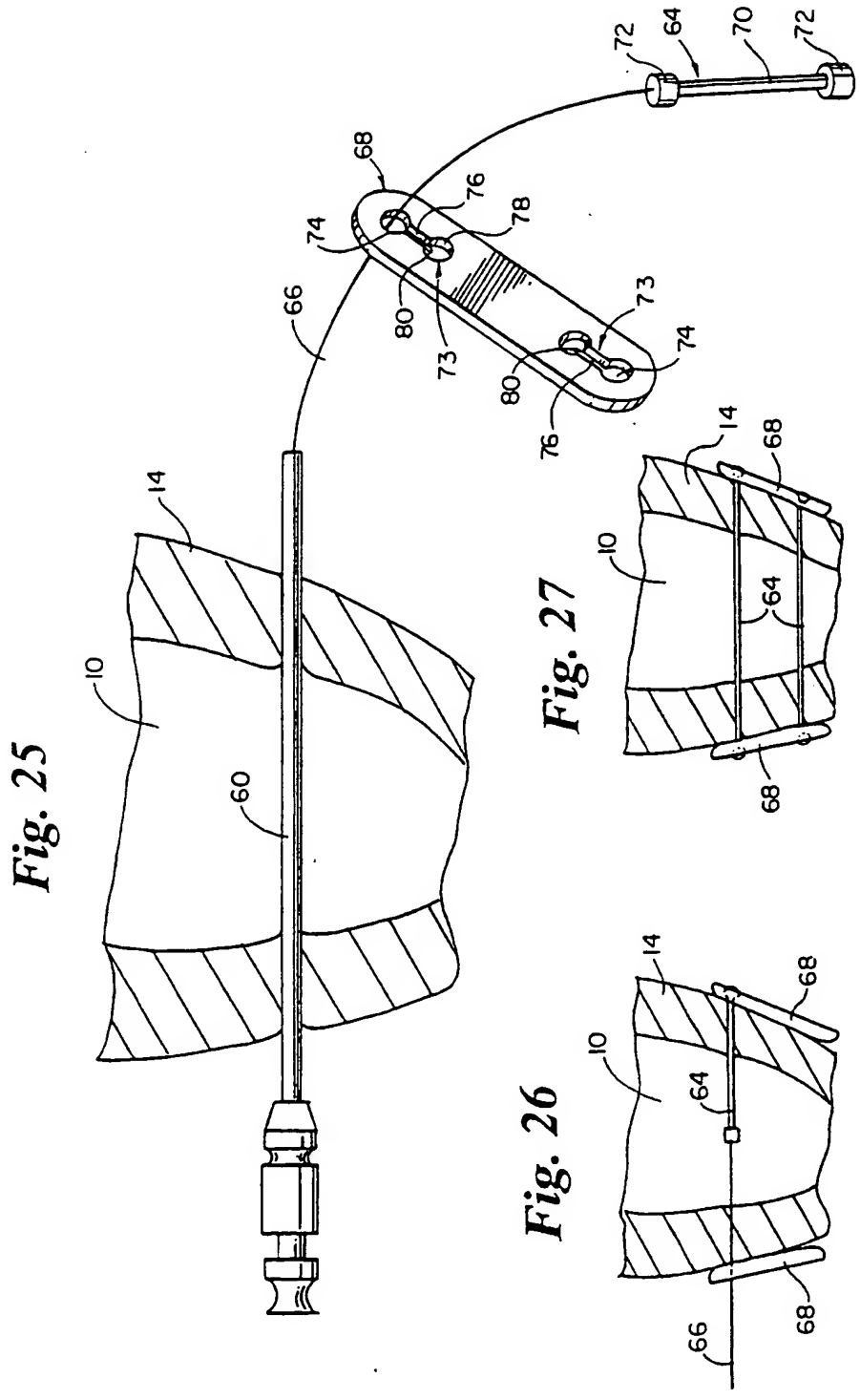


Fig. 25

Fig. 27

Fig. 26

Fig. 28

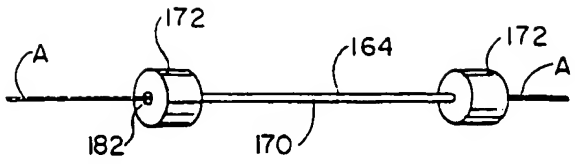


Fig. 29

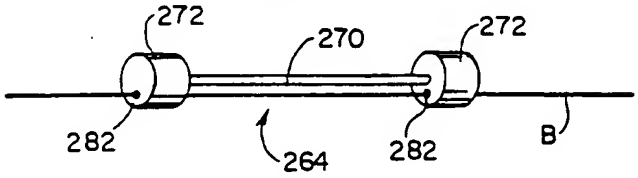


Fig. 30

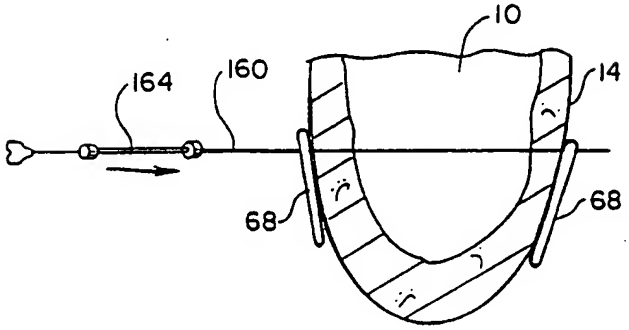


Fig. 31

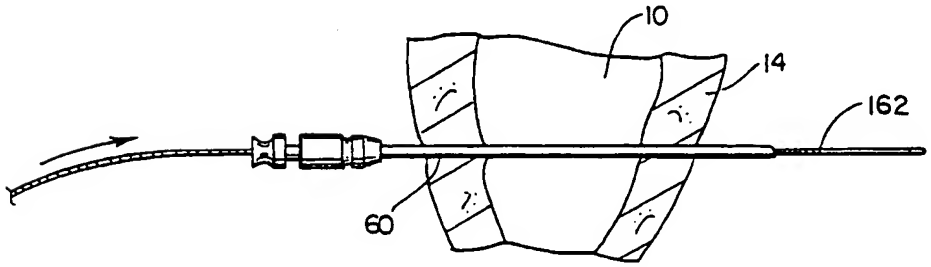


Fig. 32

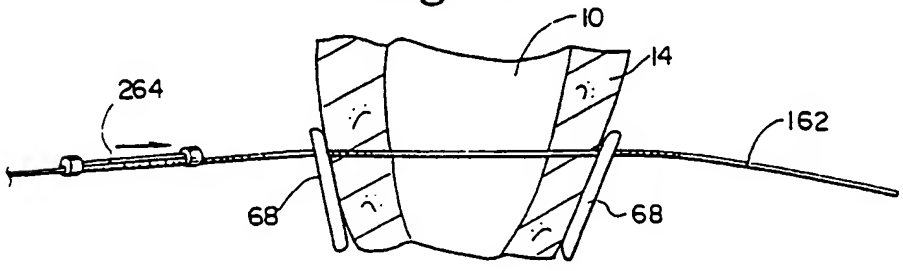


Fig. 33

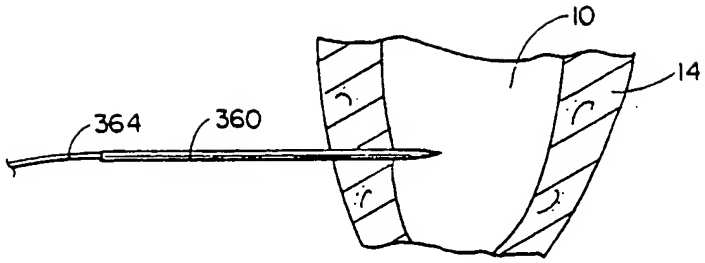


Fig. 34

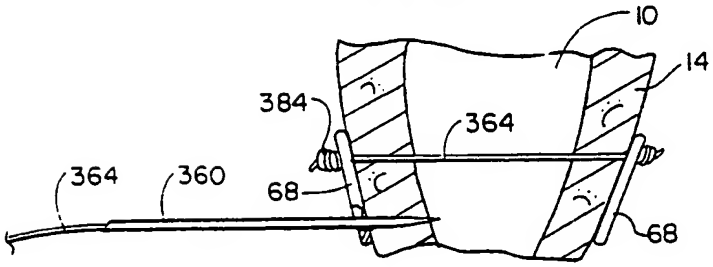


Fig. 35

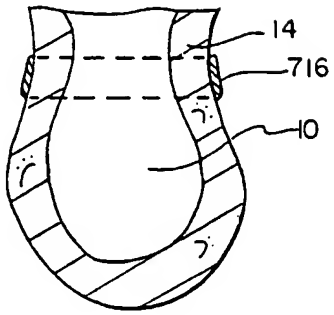
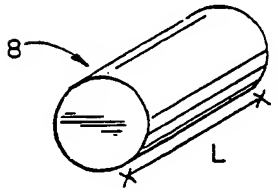


Fig. 36



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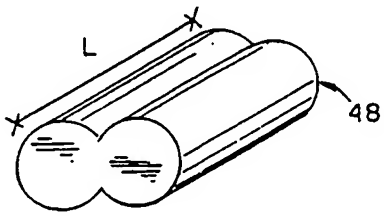

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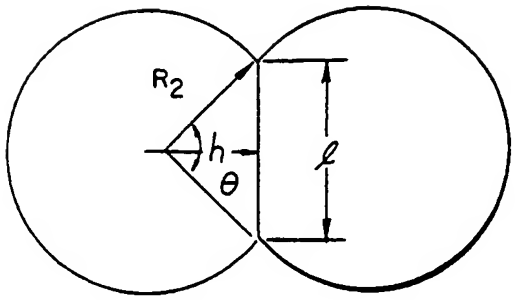

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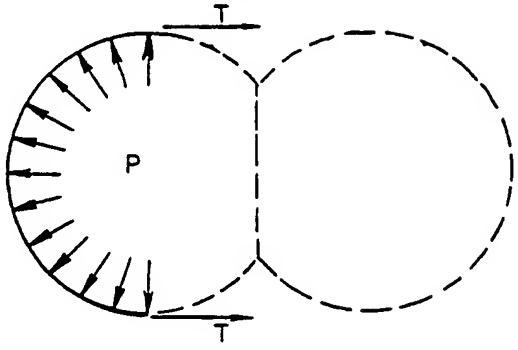
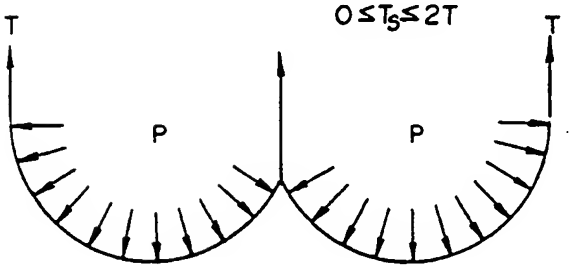


Fig. 40



INTERNATIONAL SEARCH REPORT

International application No.
PCT/US97/24116

A. CLASSIFICATION OF SUBJECT MATTER
IPC(6) :A61B 17/12
US CL :600/016
According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)

U.S. : 128/897; 600/016-018, 037; 601/011, 153; 623/003, 011

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

Electronic data base consulted during the international search (name of data base and, where practicable, search terms used)

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category*	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
X	US 5,192,314 A (DASKALAKIS) 09 March 1993, entire document.	1
X,P	US 5,702,343 A (ALFERNESS) 30 December 1997, .entire document.	1

☐ Further documents are listed in the continuation of Box C. ☐ See patent family annex.

*	Special categories of cited documents:	"T"	later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention
"A"	document defining the general state of the art which is not considered to be of particular relevance	"X"	document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone
"E"	earlier document published on or after the international filing date	"Y"	document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art
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"O"	document referring to an oral disclosure, use, exhibition or other means		
"P"	document published prior to the international filing date but later than the priority date claimed		

Date of the actual completion of the international search
16 FEBRUARY 1998

Date of mailing of the international search report
09 MAR 1998

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